condition, especially absence of the optic chiasm with temporal hemianopia, makes the specimen valuable. The existence of temporal hemianopia in this case is instructive, as pressure on the chiasm, either by a tumor or from effusion, gives the same symptoms in the visual field; but in this case, the temporal hemianopia was recognized fully a year before symptoms of compression came on, after which, death ensued in three to four weeks. The fluid within the cranial cavity was estimated at three ounces. That there was no compression in the earlier stages of the meningitis, which was chronic, is impossible to say, but it is doubtful.

A longitudinal section of one optic nerve and a cross section of the other, is to be made at the point where an attempt at forming the chiasm exists; a description of the microscopical appearances of the fibres of the optic nerves will be submitted to the Society at another time.

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## SOME REMARKS ON ASTHENOPIA AND THE CHANGES IN REFRACTION IN ADOLESCENT AND ADULT EYES.

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IN taking up for discussion a subject so well worn and threadbare, I must ask the indulgence of the Society, if in an attempt to emphasize what appear to me important convictions, I repeat to some extent facts and statements with which we are all familiar. I shall, however, be brief, and hope thus to avoid producing in your minds impressions similar to the mental shudder which comes over us when after a long morning's work we face "another refraction case," or to the sigh of relief with which we at last jot down the correction ordered for our patient. Let us consider for a moment the normal growth of the eyeball, how, starting in the new-born child with an average visual axis of 17.5342 mm., we find in the

average adult eye, not much used for near work, an average axis of 24.3037 mm.[1]

NOTE.—As is well known Listing has assumed for the average emmetropic eye a visual axis of 22.647 mm., while Helmholtz, at first assuming an axis of 22.231, has later adopted 22.834 as the standard. These measurements are from the anterior pole of the eye (outer surface of the cornea) to the fovea centralis. Allowing (with Donders) 1.3 mm. for the thickness of the sclerotic, we obtain in the first case 23.947 mm., in the latter 24.134 mm. as the diameter of the eyeball in its visual axis. Arlt[2] considers an eye with a corneal radius of 7.6 mm. and an axis of 24 mm. as emmetropic, and anything over 26 mm. as certainly myopic. Mauthner,[3] after a careful discussion of the subject, assumes considerable variation in the axis of emmetropic eyes, which may either have a sharply curved cornea with correspondingly shorter diameter of the eyeball, or a less curved cornea with greater diameter of eyeball. As extremes, he states that we may have an emmetropic eye with a corneal radius of 6.95 mm. and a visual axis of 20.95 mm., or a corneal radius of 8.04 mm. with an axis of 24.94 mm. These measurements are from the outside of the cornea to the fovea, and if we add 1.3 mm. for the thickness of the sclerotic we obtain 22.25 mm. as the diameter of the smaller eyeball, and 26.24 mm. as that of the larger eyeball. The same author reckons the axis of various grades of hypermetropia as follows (Ibid. p. 642):

| | | | | | |
|---|---|---|---|---|---|
| $H = \frac{1}{40}$, | Corneal Radius | 7.77, | diameter in visual axis | | 24.70. |
| $H = \frac{1}{12}$ | " " | 7.62, | " " | " | 23.67. |
| $H = \frac{1}{6}$ | " " | 7.48, | " " | " | 22.56. |
| $H = \frac{1}{4.5}$ | " " | 7.43, | " " | " | 21.85. |

If the eye remained only partially developed it would, even with an hypermetropia of $\frac{1}{4.5}$, have increased to 20.55 mm., while a hypermetropia of $\frac{1}{12}$ would correspond to an axis of 22.30 mm., and a hypermetropia of $\frac{1}{40}$ would give us an axis of 23.40 mm. Thus, even for the very highest grades of hypermetropia, the eye would have grown 3 mm., while to reach emmetropia it must have increased at least 6.5 mm. in its visual axis. When we look at the soft, elastic, bluish sclerotic of the young, we can readily appreciate, how under any softening influence it would give way slowly, even to normal intraocular pressure. Transient congestion is the universal law of physio-

[1] Jaeger, Einstellungen des dioptrischen Apparats. Wien, 1861, p. 14.
[2] Ueber die Ursachen und Entstehung der Kurzsichtigkeit, p. 2. Wien, 1876.
[3] Mauthner, Uber die optischen Fehler des Auges, p. 423. Wien, 1876.

logical activity; if we use our muscles more blood circulates in them, and for the time they become tense and swollen. If we irritate the stomach by food, it becomes red; and if we converge and accommodate with our eyes, more blood flows to them, the vast choroidal vascular network, as well as the ciliary processes and ciliary muscle, occupy more space and transiently raise the intraocular tension, while the sclerotic also carries more blood, and its lymph spaces, as well as those of the cornea, become fuller of serum. The retina shares in the general excitement; it becomes swollen and less transparent, and presents a faint pinkish hue in its thicker parts, it vails but does not entirely hide the upper and lower margins of the disc. The walls of the larger blood-vessels at or near their emergence from the disc become visible, and numerous yellowish or silvery reflexes may be seen in various parts of the eye-ground, near the smaller vessels, and are probably due either to a similar cause, or to an alteration of their lymph sheaths. We have in short a state which has been so well described and pictured by Jaeger as retinal irritation. (*Netzhaut Reizung.*)

In healthy individuals where there are sufficient intervals of rest, the blood-vessels contract, the increased exudation of serum is absorbed, and the eye once more subsides into a state of quiescence. If, however, the period of work be too long prolonged or too often repeated, the capillaries remain dilated, the lymph spaces fuller, and there is never a period of complete contraction and quiet. The reflex actions which determine a normal flow of blood to the organ become disturbed, and we have a congestion of the eyelids and of the tarsal and bulbar conjunctiva, accompanied by an itching, burning, and watering of the eye, with a feeling of sandiness or sleepiness. Whenever the exertion is continued and pushed still further, the weary muscles ache, and we have pain in and back of the eye, and also shoots through the temple and forehead. Frequent repetitions of this process cause the congested, softened, and serum-infiltrated tissues to give way even under the *normal* intraocular pressure. This yielding usually takes place at the posterior pole, where the sclerotic is best supplied with blood, but

where it is at the same time most weakened by the numerous perforations in its coats, due to the entrance of the posterior ciliary arteries and nerves.

Any constitutional dyscrasia which makes the circulation sluggish and the tisses less tense and resisting—notably those morbid processes which are usually grouped under the name of scrofula—greatly augments this tendency to change, as does also the constitutional feebleness ensuing after eruptive and continued fevers. Any inflammation of the choroid may also cause softening and cause the eye to give way to its normal intraocular pressure on the slightest attempt at near work, thus producing the myopia which we not unfrequently see among unlettered people. Although not usually classed with myopia, I cannot but regard conical cornea and its cogeners, the high grades of mixed astigmatism, as usually examples of similar congestive softenings, affecting the anterior parts of the eye; although they may have had their starting point in strumous or other local lesions. These defects, like true myopia, are increased by persistent attempts at near work. With this summary of my views as regards the congestive softenings of the eyeball, the diminution of hypermetropia and its passage into myopia, we may now study a little more minutely the asthenopic symptoms which accompany these changes.

The low grades of congestion of the tarsal and ocular conjunctiva cause the eye to feel sandy and the lids heavy, while the individual becomes drowsy and perhaps falls to sleep promptly over his book. When eye-work is persisted in after this point, we have blurring of the print, and burning and itching of the eyes. If work still be persisted in, neuralgic pain develops, and is felt in the eye itself, but is usually more marked in the forehead and temple, while in some cases there are shoots of pain back to the junction of the head and neck. If such eyes are examined with the ophthalmoscope ample evidence is obtained that there is a congestion of the retina and of the intraocular end of the optic nerve, as well as of the conjunctiva and lids.

We find the retinal fibres around the disk infiltrated and swollen, causing its pink to be more or less mixed with gray.

The outlines of the nerve are clouded, and at times invisible, especially in the neighborhood of the larger vessels. The swelling is often sufficient to cause the disc to become prominent, while the choroid shows the disturbance of its circulation by an increase in its granular aspect, each cell or perhaps each group of cells (corresponding to a capillary loop), swelling so as to cause a slight elevation of its surface. When this state goes further and the pigment is disturbed in its distribution, the eye-ground looks black-peppered, and honeycombed. In this state of the eye-ground, the walls of the larger vessels change their index of refraction and therefore often become visible on the disc, and to considerable distances from it. The lymph sheaths which accompany and surround the retinal vessels may often also be seen as gray or whitish streaks along the vessels, and as silvery reflexes in a level with them or in front of them.

Almost all cases of myopia will tell you that there was a time when they had considerable neuralgic pain in the forehead, temples and eyes, as well as itching and burning on the prolonged use of the eyes, but that while the latter symptoms still often recur, the neuralgia has disappeared. This is due to the fact that the neuralgia is present so long as the ocular shell is tough and resistant, but usually disappears when it softens and gives way.

In striking contrast to this form of asthenopia is that occurring in medium grades of hypermetropia (*e. g.* from $\frac{1}{8} - \frac{1}{18}$), where the sclerotic is dense, thick and unyielding, and where severe neuralgic pain is often the prominent symptom of the asthenopia. The more mobile and weaker the nervous system of the patient, the more severe are the neuralgic pains. The eyes often become so sensitive that any rapid change of focus from distant to near objects is uncomfortable, and is in some cases accompanied by nausea and dizziness. Such eyes usually do not become myopic (although the following table shows a rapidly diminishing hypermetropia of $\frac{1}{12}$), the severe discomfort consequent upon their use either forcing the patient to to wear a correcting glass, or causing him to limit his work or change his occupation.

Probably, also, the greater thickness of the sclerotic, and the fact first pointed out by Arlt, that owing to the small size of the eyeball it is not possible for the external rectus during convergence to press upon the corresponding vortex vein, and thus augment intraocular congestion, may go far to prevent any lengthening of the visual axis.

Nothing to my mind demonstrates more forcibly the truth of the foregoing remarks than the prompt relief of many cases of long standing and constantly recurring conjunctivitis and blepharitis by the habitual use of correcting glasses. The cases of diminishing hypermetropia of low grade, which become stationary under the use of correcting glasses, and in which eyes previously asthenopic become able to perform their usual work with comfort, point also in the same direction. Similar cases must have been observed by many of my fellow members; but notwithstanding, how frequently do we hear that it is not worth while to correct low grades of ametropia, and how often do we see that such cases, after extensive courses of nitrate of silver, zinc, alum, douches, etc., find relief in the rest which follows the use of a mydriatic and proper glasses. It is my belief that it is always worth while to correct ametropia, no matter how slight, if it produces asthenopia, and that we thus go far to prevent congestion and softening of the eye and its elongation in the visual axis. There is another large group of cases, where, even with a considerable degree of astigmatism or hypermetropia, glasses fail to cure the neuralgia and discomfort, although even here they frequently alleviate the suffering and materially augment the working powers of the patient.

There are cases where the ametropia occurs in individuals affected with disease or perverted function of other important organs. In the male sex we often find examples in incipient tabes dorsalis or in meningeal irritation, while in women they are very frequently associated with disease of the uterus and ovaries, or with perverted action of these organs, such as frequently occurs during their development in the young or their shrinking in the old. In such cases very slight optical defects are accompanied by severe neuralgia, not only in the

eye, forehead and temple, but also in the top of the head and at the juncture of the head with the neck; at times even shooting down the spine. Such symptoms often come at unusual times, especially on waking in the morning, when the eyes, instead of being refreshed by sleep, are sandy and painful and unable to bear the light. The patients often complain of the lids sticking to the eyeball during sleep, so that on waking during the night, they have difficulty in opening their eyes even after resorting to bathing them in warm water. This is due to perverted sensation, and not to any glueing of the conjunctival surfaces by secretion. Of course all such cases receive only partial relief from glasses, but they nevertheless often derive great comfort from them, and are thus enabled to do a considerable amount of work with less discomfort than they previously suffered. Where the ametropia is but slight, such patients, when their health has been re-established, are often able to lay aside the weak cylinders or convex glasses which have previously afforded them so much comfort. The same ability to lay aside glasses previously necessary is also often seen after convalescence from acute disease, when slight optical defects, which previously passed unnoticed, become unbearable, and where the discomfort gradually vanishes as they regain their health.

To emphasize and maintain these views, I have selected from my case books and hospital records, a few instances, all of which have been carefully studied for years, and all of which have been repeatedly atropinized. To facilitate ready reference and comparison, they have been arranged in two tables, —First, Diminution of Hypermetropia (six cases); second, Passage of Hypermetropia into Myopia (five cases).

## ANALYSIS OF SIX CASES OF GRADUAL DIMINUTION OF HYPERMETROPIA,

Each determination of the Refraction in every case having been made after Absolute Paralysis of the Accommodation by the repeated use of Atropine or Hyoscyamine.

| No. of Case. | Sex. | Age at 1st examina'n. | Occupation. | Date of first Examination and State of Refraction and Acuity of Vision at that time. | | Date of second Examination, and State of Refraction and Acuity of Vision at that time. | | Date of third Examination, and State of Refraction and Acuity of Vision at that time. | | REMARKS. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F | 12 | School-Girl, subsequently Clerk. | 1874. | | 1876. | | 1881. | | In right eye, in 1874, there was a pigment loop to the outside of the disc, separated from it by a crescent of normal colored choroid, while in 1881 there was a semi-atrophic crescent. In left eye, in 1874, there was a slight brownish crescent to the outside of the disc; while in 1881, there was a semi-atrophic conus with black outer margin, and another pigment loop beyond. Insufficiency of interni, 8°. |
| | | | | R. E. | L. E. | R. E. | L. E. | R. E. | L. E. | |
| | | | | + $\frac{1}{18}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{20}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{24}$ s. ○ + $\frac{1}{48}$ c., axis 75°. V.=$\frac{20}{20}$ | + $\frac{1}{24}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{48}$ s. ○ + $\frac{1}{48}$ c., axis 90°. V.=$\frac{20}{20}$ | + $\frac{1}{48}$ s. ○ + $\frac{1}{48}$ c., axis 90°. V.=$\frac{20}{20}$ | |
| 2 | F | 16 | School-Girl. | 1873. | | 1874, about 11 mos. later. | | | | In the interval between the examinations the glasses were not worn constantly, and the patient complained of headache, itching and burning of the eyes and blurring of the print. |
| | | | | R. E. | L. E. | R. E. | L. E. | | | |
| | | | | + $\frac{1}{24}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{30}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{42}$ c., axis 90°. V.=$\frac{20}{20}$ | + $\frac{1}{60}$ s. ○ + $\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | | | |
| 3 | M | 10 | Schoolboy, later College Student. | 1872. | | 1877. | | 1881. | | Pain back of the eyes, congestion of the ocular conjunctivæ and of the margins of the eyelids. At the two earlier dates severe neuralgic pain in temple and forehead. At the latter, no more acute pain. Ophthalmoscope shows great haziness of the retina, and wooliness of the choroid. |
| | | | | R. E. | L. E. | R. E. | L. E. | R. E. | L. E. | |
| | | | | + $\frac{1}{42}$ s. V.=$\frac{20}{20}$ ($\frac{20}{15}$?) | + $\frac{1}{42}$ s. V.=$\frac{20}{20}$ ($\frac{20}{15}$?) | + $\frac{1}{42}$ c., axis 90°. V.=$\frac{20}{20}$ | + $\frac{1}{60}$ s. ○ + $\frac{1}{60}$ c., axis 60°. V.=$\frac{20}{20}$ | + $\frac{1}{144}$ s. ○ + $\frac{1}{144}$ c., axis 90°. V.=$\frac{20}{20}$ with difficulty. | + $\frac{1}{144}$ s. ○ + $\frac{1}{144}$ c., axis 60°. V.=$\frac{20}{20}$ with difficulty. | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 4 | M | 25 | Student of Theology, later Clergyman. | 1874. | | 1885. | |
| | | | | R. E. | L. E. | R. E. | L. E. |
| | | | | + $\frac{1}{36}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{42}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{72}$ s. ⊂ + $\frac{1}{72}$ c., axis 80°. V.=$\frac{20}{20}$ | + $\frac{1}{72}$ s. ⊂ + $\frac{1}{72}$ c., axis 90°. V.=$\frac{20}{20}$ |
| 5 | M | 30 | Clerk. | 1872. | | 1886. | |
| | | | | R. E. | L. E. | R. E. | L. E. |
| | | | | + $\frac{1}{36}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{36}$ s. V.=$\frac{20}{20}$ | + $\frac{1}{48}$ c., axis 60°. V.=$\frac{20}{20}$ | + $1\frac{1}{44}$ s. ⊂ + $1\frac{1}{44}$ c., axis 30°. V.=$\frac{20}{20}$ |
| 6 | F | 14 | School Girl. | 1877. | | 1884. | |
| | | | | R. E. | L. E. | R. E. | L. E. |
| | | | | + $\frac{1}{12}$ s. ⊂ + $\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | + $\frac{1}{12}$ s. ⊂ + $\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | + $\frac{1}{24}$ s. ⊂ + $\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | + $\frac{1}{18}$ s. ⊂ + $\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ |

Insufficiency of recti interni.

Marked blepharitis and conjunctivitis, with great sensitiveness to artificial light. Grayish nerves.

The astigmatism remaining the same, we have an increase in the refraction of one twenty-fourth in the right eye, and of one thirty-sixth in the left.

## ANALYSIS OF FIVE CASES OF THE PASSAGE OF HYPERMETROPIA INTO MYOPIA,

Each determination of the Refraction having been made after Absolute Paralysis of the Accommodation by the repeated use of Atropine or Hyoscyamine.

| No. of Case. | Sex. | Age at 1st Exam'n. | Occupa'n. | Date of first Examination, and State of Refraction and Acuity of Vision at that time. | | Date of second Examination, and State of Refraction and Acuity of Vision at that time. | | REMARKS. |
|---|---|---|---|---|---|---|---|---|
| | | | | 1881. | | 1886. | | |
| | | | | R. E. | L. E. | R. E. | L. E. | |
| 1 | M | 11 | School Boy. | $+\frac{1}{60}$ s. $\bigcirc$ $+\frac{1}{60}$ c., axis 105°. V.=$\frac{20}{30}$ | $+\frac{1}{42}$ s. $\bigcirc$ $+\frac{1}{36}$ c., axis 105°. V=$\frac{20}{30}$ | $-\frac{1}{30}$ s. $\bigcirc$ $-\frac{1}{20}$ c., axis 15°. V.=$\frac{20}{20}$ | $-\frac{1}{60}$ c., axis 180° $\bigcirc$ $+\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{30}$ | |
| | | | | 1881. | | 1883. | | |
| | | | | R. E. | L. E. | R. E. | L. E. | |
| 2 | F | 10 | School Girl. | $+\frac{1}{60}$ c., axis 180°. V.=$\frac{20}{20}$ | $-\frac{1}{60}$ c., axis 60°. V.=$\frac{20}{20}$ | $-\frac{1}{24}$ s. $\bigcirc$ $-\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | $-\frac{1}{24}$ s. $\bigcirc$ $-\frac{1}{48}$ c., axis 90°. V.=$\frac{20}{20}$ | Passage of $A^h$ in one eye into M+$A^m$, the degree of $A^s$ remaining unaltered; of $A^m$ in the other to M+$A^m$. Patient a frail child, with hereditary tendency to phthisis. |
| | | | | 1874. | | 1885. | | |
| | | | | R. E. | L. E. | R. E. | L. E. | |
| 3 | M | 20 | Clerk. | $+\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | $+\frac{1}{60}$ c., axis 90°. V.=$\frac{20}{20}$ | $-\frac{1}{48}$ c., axis 180°. V.=$\frac{20}{20}$ | $-\frac{1}{42}$ c., axis 165°. V.=$\frac{20}{20}$ | In right eye, in 1874, there was a slight pigment loop to the outside of the disc, but in 1885 there was a semi-atrophic conus as wide as the largest part of the main retinal vein. In left eye, in 1874, slight absorbing conus with heavy pigment border at the outside. In 1885, an annular conus with faint black sprinkling in places, but the marking of pigment to the outside has entirely disappeared. |
| | | | | 1877. | | 1882. | | |
| | | | | R. E. | L. E. | R. E. | L. E. | |
| 4 | M | 22 | Law Student. | $+\frac{1}{48}$ c., axis 90°. V.=$\frac{20}{20}$ | $+\frac{1}{60}$ c., axis 85°. V.=$\frac{20}{20}$ | $-\frac{1}{60}$ c., axis 180°. V.=$\frac{20}{20}$ | $-\frac{1}{60}$ c., axis 150°. V.=$\frac{20}{20}$ | We have here an almost regular spheroidal dilatation of the eye—the emmetropic meridian having become slightly myopic, and the hypermetropic meridian having changed to an equal degree in the left eye, thus making it emmetropic. |
| | | | | 1877. | | 1882. | | |
| | | | | R. E. | L. E. | R. E. | L. E. | |
| 5 | M | 17 | College Student. | $+\frac{1}{48}$ c., axis 180°. V.=$\frac{20}{20}$ | $+\frac{1}{48}$ c., axis 90°. V.=$\frac{20}{20}$ | $-\frac{1}{20}$ s. V.=$\frac{20}{20}$ | $-\frac{1}{24}$ s. $\bigcirc$ $-\frac{1}{48}$ c., axis 180°. V.=$\frac{20}{20}$ | Cases 4 and 5 were brothers—their father highly myopic, their mother with nearly normal vision. The right eye has lost its astigmatism and become myopic in all meridians alike. In the left eye, the amount of astigmatism remains the same, and the meridian of strongest curvature retains the same direction, while the refraction has increased one-sixteenth in all meridians. |

In conclusion :—

I.—It must be remembered that a diminishing hypermetropia and an increasing myopia are merely arbitrary expressions referring to different stages in a process essentially the same; in both cases it being simply a slow distension of the softened eyeball in the direction of its visual axis.

II.—I would urge therefore the careful correction of every case of ametropia where there is any accompanying asthenopia; such correction going far to diminish and prevent the congestive and softening influences which always accompany the use of the eye and facilitate the diminution of hypermetropia and the production of myopia. In my judgment, this is quite as important a preventive of elongation of the visual axis, as any other one measure to which we habitually resort, not excepting the usual precautions as to lighting, hygiene, and health.

III.—All such cases should be carefully measured under a strong mydriatic, as it is impossible to correct them accurately without it, either by the use of test glasses, of the ophthalmoscope, or of retinoscopy. Moreover, the enforced rest of the eye during the influence of the mydriatic, is an important aid in diminishing its congestion.

IV.—We should carefully watch all cases of hypermetropia where the ophthalmoscope shows a decided difference of level between the disc and the macula. They are usually cases of slow distension of the eyeball and diminishing hypermetropia. The same rule holds good in cases of increasing myopia.

V.—During the dilatation of the pupil by the mydriatic, we should insist on our patients wearing dark glasses, even in a moderate light. The immense quantity of light otherwise admitted to the eye is likely to irritate it, and at times may cause a low grade of chorio-retinitis, which it is much easier to call into existence than to cure.

DISCUSSION.

DR. THEOBALD.—It strikes me that there must have been in many of these cases not only a change in the absolute refraction, but an increase of the astigmatism. A change in the strength of the cylinders was required. It will be remembered that last year I reported three cases of what I regarded as progressive astigmatism. In two of these, during a series of years, the astigmatism had apparently doubled in amount. Since I presented these cases, I have had some correspondence with Dr. Randall, who has almost persuaded me that some of them were not genuine instances of increase of the astigmatism, but rather the result of asymetrical action of the ciliary muscle. Under the use of cylinders, the muscle had yielded and allowed more of the astigmatism to come to the surface. This suggests itself in Dr. Norris' cases. If, however, there were such an asymetrical action of the muscle, we should expect that in a myopic patient the relaxation of the ciliary muscle would produce a diminution of the myopia. In the cases reported, the change was in the opposite direction; as the astigmatism increased the myopia developed. I should like to hear Dr. Norris' views in regard to this decided increase of astigmatism.

DR. HARLAN.—No doubt, in nearly all cases, changes in the refraction, particularly spherical changes, are due to changes in the axis of the eye. As has been suggested, there are some which can be referred to changes in the cornea. I recently had the opportunity of examining a patient for whom I prescribed cylindrical glasses several years ago. When examined in 1878, the patient was thirty-one years of age, a theological student. I used a strong solution of atropia, repeated frequently, and assured myself that the ciliary muscle was paralyzed. At that time, under atropia, correction with a + 1.5 D. cylinder in each eye, axis at 90°, gave V. = $\frac{20}{20}$. This answered very well until lately. He now complained of asthenopia, and said that distant vision was not good. I again atropinized the eyes, and found that the low degree of simple hypermetropic astigmatism had changed to mixed astigmatism. In the right eye — 1. D. spherical with + 3. D. cylindrical, gave V. = $\frac{20}{20}$, in the left eye $\frac{20}{15}$. The ophthalmoscope showed nothing abnormal. I am certain that there was no action of the ciliary muscle at the time of either examination.

DR. GRUENING.—I was specially interested in one class of cases which Dr. Norris mentioned. The patient arises in the morning with a pain in the eyes; at times he complains of his eyes being sandy; after an hour or two the eyes feel better.

There is also some photophobia. Such patients are not able to fix an object for any length of time, and often have lachrymation. This peculiar class of cases can not be corrected with glasses. I have described these cases in a paper on reflex ocular symptoms in nasal disease. Looking into the nose, I find evidence of nasal disease. The nasal reflex plays an important part in our modern pathology. All affections depending upon nasal reflex are increased by the recumbent posture, as this favors congestion of the erectile tissue. I find that these patients are better when they are up and about.

If eyes which have rested all night manifest painful sensations in the morning, we may exclude ordinary asthenopia. I found in these cases considerable swelling of the lower borders of the turbinated bones. This was treated by ordinary anti-catarrhal remedies or by the actual cautery, and, in some cases, the diseased tissue was removed with the snare. In other cases there was stenosis, due to cartilaginous excrescences, which were removed. Of a series of two hundred cases thus treated, I have certainly benefited one hundred and fifty. The remaining fifty disappeared, because they were so much startled by the treatment proposed.

Dr. Risley.—It is probably true that certain eye troubles, as described by Dr. Gruening, may be set up by pathological states in the nasal passages, but I am sure that nasal troubles do not account for the slowly distending eye-balls to which Dr. Norris has referred in his paper. I became much interested in the early history of myopia and myopic astigmatism during an examination of the eyes of the school children in Philadelphia, which I made in 1878–81. Prior to the commencement of that work I suspected, and during its progress was convinced, that the myopic eyes were not recruited from the emmetropic eyes, but from those presenting hypermetropic refraction and principally hypermetropic astigmatism. This opinion, advanced in the report of that work, published in 1881, was based upon, first, a group of cases in which hypermetropic refraction had been seen to pass into myopic refraction, and second, upon the fact that the eyes with hypermetropic astigmatism presented a greater percentage of the same pathological appearances, which in their later stages were confessedly characteristic of progressive myopia, viz., choroiditis and posterior staphylomata. Since the publication of the school report, two additional groups of cases—in which *hypermetropic* astigmatism had been observed to pass over into *myopic* astigmatism—have been brought forward, as corroborating the claims set forth in the report of the school work. As the

subject has been studied under a steadily widening experience, I have been more and more thoroughly convinced of the correctness of the views which Dr. Norris has expressed in his paper, that disturbance of the nutrition of the eye-ball, consequent upon the eye strain in hypermetropic refraction, leads to its softening, and thus permits its distension under the strain of protracted near work.

I was much impressed by his remark referring to the anterior distension, making conical cornea and posterior staphyloma dependent upon the same condition, an opinion which I have long entertained. As illustrating this view, I will refer to a case already published in the second group of cases of changed refraction. A young woman, first seen in 1874, suffering from asthenopia, after prolonged use of atropia selected a convex sphero-cylindrical glass for each eye. During the subsequent years marked change in the refraction of both eyes occurred, without the usual changes in the region of the optic nerves. At the present time the hypermetropia has disappeared in the left eye, leaving a low grade of myopic astigmatism. In the right eye, during the depression following the protracted nursing of her invalid mother, the vision sunk to $\frac{1}{20}$, consequent upon conicity of the cornea. She at the same time complained of her severe and constant headache. Under the continued use of mydriatics alternating with eserine and a pressure bandage, the curvatures of the cornea became more regular, and finally it became possible to correct them. A + 1. spherical with a — 5. cylinder, axis at 100°, gave V. = $\frac{20}{30}$. There was no further trouble until two weeks ago, when she came to the office with the eye paining her again. The glass had been broken and had been put in with the axis ten degrees out of the proper plane. After the use of homatropine for several days, she selected the same glass, and it gave the same vision. This case bears out the view expressed. An eye-ball which had been slowly distending, suddenly, from the general softening of the tissues due to depressed health, rapidly distended in an anterior direction. It has fallen to my lot a number of times to watch cases of progressive near-sight, in which the characteristic changes were not seen around the optic nerve, but in which the anterior perforating vessels were dilated and the scleral region obviously thin, and the myopia increasing. In a large number of cases of myopia the distension may be more or less regular anteriorly. I am convinced that certain cases of conical cornea are analogous to posterior staphyloma in progressive myopia.

DR. RANDALL.—Of the many wide questions as to the causation of myopia suggested by the paper just read, there is one which I would like to bring to the attention of the Society, in hope that evidence positive or negative can be given by members that will aid in its solution. The proposition is best set forward and in the least equivocal form by Stilling, in the Archives of Ophthalmology, who considers all cases in one of three classes. First, myopia due to excessive corneal curvature, or a physiological myopia which is the adaptation of the eye to its environment—a growth which is a form of evolution, and is a purely physiological adaptation. Beyond this he places a class due to strain and overwork; and adds further a category of excessively high grades of what he calls pernicious myopia, where the eye, from serious choroidal change, assumes a high degree of myopia. I have been struck by the assumption by some other authors of the great importance of this matter of physiological growth in the causation of myopia. In the cases which I have studied, there has seemed a distinct pathological increase in the axis of the eye, accompanied by pathological changes such as choroidal disturbance, and by asthenopia. I would ask what ground is there for an assumption like that made by Dor,[1] when he states that we can recognize in the cradle the eye which is physiologically myopic, although the false science of modern times would call it emmetropic or hypermetropic, because at present rays of light are not focused in front of the retina?

DR. NORRIS.—I would say with reference to the remarks of Dr. Theobald, that I have purposely sorted out the cases which could in any way be attributed to corneal change, and have included those which I believe are due to axial change. I have a number of cases of change in the anterior part of the eye, cases of mixed astigmatism changing their refraction, due to congestive softening of the anterior part of the eye. I have not considered these to-night, but have contented myself with the cases which I have given, in which there was no change in the anterior part of the eye, but which appeared to be due to distention of the eye-ball in the macular region.

Only two of the cases show any considerable increase in the amount of astigmatism; in the others there appears to have been a spheroidal increase in the diameter of the eye-ball. To take in all these questions would require a large amount of time, and I do not know that I have thoroughly worked them out. I am rather disposed to think that astigmatism is usually

[1] "Etude sur l'Hygiène Oculaire au Lycée de Lyon," p 12. Paris, 1878.

due to some change in the curvature of the cornea or of the lens, and can only regret that I have no ophthalmometric measurements of these cases. They show, for instance, that hypermetropic astigmatism starting at $\frac{1}{48}$ will pass over to myopic astigmatism of equal amount in ten years. In one or two of the cases, I am quite sure from the amount of choroidal change that the change of refraction must have been due to distention of the posterior part of the globe.

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## ON THE COMBINATION OF TWO CYLINDRICAL LENSES.[1]

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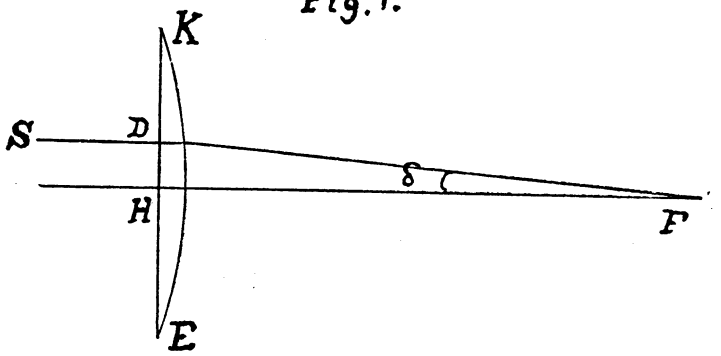
Fig. 1.




LET K E, Fig. 1, represent a section of a thin plano-cyl. lens made by a plane perpendicular to the cylinder-axis, the lines of the diagram being supposed to lie in the plane of the section.

H F is perpendicular to K E at its middle point H.

H F $= f =$ principal focal distance (neglecting thickness of lens).

[1] The writer is indebted for the essential part of this paper to Prof. Oliver, of Cornell University, who about sixteen years ago, in response to some inquiries, was kind enough to send him a communication on the subject.